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Pubertal Development Predicts Eating Behaviors in Adolescence

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Abstract

Objective—Early maturing girls are at increased risk for disordered eating. However, it is unclear if the association between puberty and disordered eating continues throughout pubertal development and if a similar association is exhibited in boys.

Method—Participants included 1340 same- and 624 opposite-sex twins from the Swedish Twin Study of Child and Adolescent Development. Pubertal development was assessed at age 13–14 with the Pubertal Development Scale. General disordered eating, measured with the Eating Disorder Inventory-2 (EDI) was assessed at age 16–17, and dieting and purging behaviors were assessed at both ages 16–17 and 19–20. We applied analysis of variance and logistic regression analyses to determine whether pubertal development in early-to-mid adolescence predicted eating disorder-related behaviors in late adolescence and young adulthood

Results—Pubertal development in early-to-mid adolescence was significantly associated with EDI scores and dieting in late adolescence. No significant association was observed between pubertal development and dieting and purging in young adulthood.

Discussion—Complex combinations of cultural and biological influences likely converge during pubertal development increasing vulnerability to disordered eating. The impact of pubertal development on disordered eating appears to be limited to the adolescent period.

Keywords

disordered eating; eating disorder; puberty; pubertal development; development; adolescence

Early puberty, as measured by either pubertal status or pubertal timing, is associated with eating disorders and disordered eating in girls.[1, 2] Pubertal status refers to stage of pubertal development and is often assessed by asking individuals to rate their perception of their pubertal development based on secondary sex characteristics such as hair growth and skin changes. This continuous measure of pubertal development is then categorized to correspond to one of five stages typically based on Tanner's classification scheme. Scores correspond to the: (1) prepubertal, (2) beginning pubertal, (3) midpubertal, (4) advanced pubertal, or (5) postpubertal stage.[3, 4] Girls in more advanced stages of pubertal development often report increased prevalence of disordered eating and weight-loss attempts than girls in less advanced stages,[5–8] and pubertal stage is more strongly associated with eating disorder symptoms than chronological age during the peripubertal period.[5] Pubertal

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status in early adolescence also predicts disordered eating one year later.[9] However, this result has not been consistent.[10, 11]

Pubertal timing refers to the age of a pubertal event (e.g., age at menarche) and is often assessed relative to a referent group in order to categorize pubertal timing as early, late, or on time.[2] Prospective and retrospective reports suggest that earlier pubertal timing is associated with elevated rates of eating disorders and bulimic-related symptomatology.[1, 12–15] However, some studies have not shown pubertal timing to significantly predict later disordered eating in girls, especially at older ages.[11, 16, 17] These discrepant results could indicate that the impact of puberty on disordered eating is constrained to a specific developmental period.[18] Puberty may only increase the risk for disordered eating during the developmental period during which it occurs (i.e., adolescence). This suggests that longitudinal studies focusing on adolescence are necessary to delineate the impact of puberty on eating disorders.[18] Moreover, it is not known whether pubertal development continues to impact the vulnerability for disordered eating across the later stages of puberty regardless of the timing of the pubertal event. Studies to date have focused exclusively on pubertal stage and timing. Simply progressing through puberty may increase risk for disordered eating.

Research examining the association between puberty and disordered eating in boys is sparse. However, studies have suggested early maturation in boys is associated with more favorable social outcomes and fewer attention problems,[19, 20] but is also related to externalizing behaviors.[1, 21, 22] For disordered eating, results are inconsistent. Cross-sectional and retrospective reports indicate that boys who report maturing earlier than peers have increased levels of disordered eating behaviors.[14, 23] In contrast, boys who report that their pubertal development was on time or late show elevated body dissatisfaction compared with boys who report maturing earlier than their peers.[15] However, three longitudinal studies suggest that pubertal status, based on the five Tanner stages of development, is not predictive of disordered eating in boys.[9–11]

Examining the association between disordered eating and pubertal timing defined as the age of a pubertal event, is difficult in boys. Unlike girls, there is no single measure of pubertal timing that is equivalent to age at menarche. However, younger ages of oigarche (onset of ejaculations) have shown an association with bulimic symptomatology.[13] There was no association between bulimic symptomatology and late or normal onset oigarche. Currently, it is unclear whether puberty presents the same association with and risk for disordered eating in boys that it does for girls.

The goal of the current report is to examine the impact of pubertal development on disordered eating in girls and boys. In this longitudinal study, we assess whether pubertal development at age 13–14, defined continuously, predicts general disordered eating, dieting, or purging behaviors at age 16–17 and dieting or purging at age 19–20. Because our prospective report spans adolescence to young adulthood, we are also able to directly test the hypothesis that the impact of pubertal development on disordered eating is constrained to the adolescent period.

Our overarching hypothesis is that a significant association will be observed between pubertal development and eating behaviors only during the period of adolescence when pubertal change is occurring and remain salient until puberty is *completely* traversed. Specifically, based on previous research, we hypothesize that higher pubertal development scores in early-to-mid adolescence (i.e., age 13–14) will significantly predict general disordered eating, dieting, and purging behaviors in late adolescence (i.e., 16–17) in girls and general disordered eating and purging behavior in boys. Although most of our sample,

especially the girls, may be categorically classified as advanced or postpubertal by late adolescence, normal pubertal development (i.e., pubic hair growth, height, breast development) can continue well into age 16 for girls and age 17 for boys.[3,4] Moreover, using a continuous measure for puberty allows us to capture subtle differences in advanced development that are difficult to capture using a classification schedule. Finally, we do not expect to find a significant association between pubertal development and eating behaviors in young adulthood (i.e., age 19–20) when most participants would have *completely* traversed puberty if, indeed, the impact of pubertal development on disordered eating is limited to adolescence.

METHODS

Participants

Participants were from the Swedish Twin Study of Child and Adolescent Development (TCHAD).[24] Briefly, twins born between May 1985 and December 1986 were contacted through the Swedish Medical Birth Registry. Identified twins and their parents were mailed self-report questionnaires. Four assessment waves have been conducted and we utilized information from Waves 2–4. Only those twins who participated in both Waves 2 and 3, when our primary measures of interest were first assessed, were included in the present study. Wave 2 assessed twins when they were 13–14 years-old (78% responded), Wave 3 when they were 16–17 years-old (82% responded), and Wave 4 when twins were 19–20 years-old (59% responded). Ninety-two percent of twins who responded at Wave 2 responded to Wave 3 questionnaires.[24] The final sample comprises 777 monozygotic (MZ) twins (404 girls and 373 boys), 563 dizygotic (DZ) twins (296 girls and 267 boys) and 624 opposite-sex twins. To assign zygosity, computer algorithms were applied to responses from questions about the twins' physical similarity and the frequency with which they are confused by other people. The algorithms were created from a discriminant analysis of 106 same-sex pairs where zygosity had been determined by typing 16 polymorphic DNA markers.[24]

The Ethics Committee of the Karolinska Institute, Stockholm, Sweden approved TCHAD study questionnaires. In Sweden, informed consent is implied by participants responding to and returning the assessments. This research was reviewed and approved by the University of North Carolina, Chapel Hill Institutional Review Board.

Measures

Puberty—At Wave 2, pubertal development was assessed with a self-report version of the Pubertal Development Scale (PDS).[25] The PDS characterizes secondary sex characteristics including growth spurt in height, body hair development, skin changes, breast development and menarche in girls, and facial hair growth and voice change in boys. Items are measured on a four point Likert scale: (1) development has not begun, (2) development has just started, (3) development has definitely started, and (4) development is complete. Scores range from 5–20. Menarche is coded dichotomously and rated as present (4) or absent (1). Items were summed separately for girls and boys (including menarche for girls). To ensure an accurate representation of the participant's pubertal development, participants were coded missing if any of the PDS items were missing.

Pubertal development was assessed as a continuous measure and not classified by Tanner stage for two reasons, both of which better address the question of whether pubertal development (and progression) in general increases risk for disordered eating. First, using a continuous measure allows us to better examine our hypothesis that the impact of pubertal development on disordered eating is constrained to adolescence by allowing for subtle

differences between individuals in more advanced pubertal stages to be captured. Additionally, we are able to capture subtle differences between all participants who may be classified in the same pubertal stage. However, to further examine whether the association between pubertal development and disordered eating is constrained to adolescence we explored whether late maturers (i.e., participants classified as pre- or early-puberty at age 13–14 according to Tanner staging) showed a differential association with disordered eating during mid-to-early adolescence and young adulthood compared with individuals who matured early or on time.

Disordered eating—Disordered eating was assessed at Wave 3 using the Eating Disorder Inventory-2 (EDI),[26] which has been translated and validated on a Swedish female population.[27, 28] The EDI was scored according to the EDI manual[26] and for the present report the Eating Disorder Risk Composite (EDRC) was used which is the sum of the Drive for Thinness (DT; i.e. excessive concern with dieting, preoccupation with weight and an extreme pursuit of thinness), Bulimia (B; i.e. tendency toward episodes of binge eating that may be followed with the impulse to induce vomiting), and Body Dissatisfaction (BD; i.e. belief that specific parts of the body are too large) subscales. Participants who responded to less than 75% of the total items were coded as missing. If participants had missing items but responded to more than 75% of the items, the mean for the missing item was imputed. Due to a positive skew, the EDRC was log transformed prior to analysis.

Additionally, eating disorder diagnoses were obtained from the Swedish National Patient Register. This register includes individuals who received inpatient, outpatient, or psychiatric care from both private and public sectors. For Wave 3, anyone in our sample receiving an International Classification of Diseases diagnostic code of anorexia nervosa, atypical anorexia nervosa, bulimia nervosa, atypical bulimia nervosa, other eating disorders, or eating disorder unspecified with a date of diagnosis prior to Wave 3 assessments were classified as having an eating disorder. Similarly, for Wave 4, participants were classified as having an eating disorder if the diagnosis occurred prior to Wave 4 assessments.

Dieting—Dieting behavior was examined through self-report at Waves 3 and 4: participants were asked if they have ever been on a diet. Response options included: never, once, two to four times, and five or more times. Dieting was recoded to a binary variable. Those who responded “never” did not engage in any dieting behaviors. All others were classified as having been on a diet.

Purging—Purging behaviors were also examined through self-report at Waves 3 and 4. Participants were asked if they have ever used “vomiting, purgation, or diuretics to lose or keep weight.” The response options included: never, “Yes, several times in the last three months,” “Yes, several times for at least three months,” and “Yes, have tried it some.” This variable was recoded to a binary variable. Those who responded “never” did not engage in any purging behaviors; all others were classified as having engaged in purging for weight-related reasons.

Statistical Procedures

Descriptive statistics were calculated for the eating disorder-related variables and pubertal development by sex. Differences between girls and boys were examined using analysis of variance for the EDRC and pubertal development and chi-square analysis for the dieting and purging variables.

Analysis of variance and logistic regression analyses using the PROC GENMOND procedure in SAS version 9.2[29] were used to examine the association between pubertal

development in early-to-mid adolescence and EDRC, dieting and purging in late adolescence and dieting and purging in young adulthood. Generalized estimating equations (GEE) were applied to correct for the nonindependence of the twin data. In this procedure, betas and standard errors are adjusted to account for the relatedness of the twins. Twin pairs are identified in the model by a "family number" that is shared by both members of the pair.

Within the models, the eating disorder-related variables were entered as the dependent variable and pubertal development as the independent variable. For Wave 3 analyses, eating disorder diagnosis prior to 2002, when Wave 3 assessments began, was included as a covariate to control for the presence of disordered eating prior to late adolescence. Zygosity and sex were included in the model to examine whether there were significant associations between eating behaviors and sex and zygosity. Initial models also included an interaction between pubertal development and sex and between pubertal development and zygosity to ascertain whether the analyses had to be stratified by sex or by zygosity. If the interaction terms were not significant in the initial model, the model was repeated without the interaction terms. Modeling for Wave 4 was conducted similarly: Wave 3 dieting and purging behaviors and eating disorder diagnosis prior to 2005 (when Wave 4 assessments began) were included in the model as covariates to account for disordered eating prior to young adulthood. All analyses were repeated replacing the PDS score as an independent variable with our categorical definition of puberty identifying participants as late maturers or not.

RESULTS

Sample Description

According to the Tanner classification scheme, 9% (n = 85) of boys and 1% (n = 14) of girls in this sample are classified as prepubertal, 42% (n = 399) of boys and 2% (n = 23) of girls are in the beginning pubertal stage, 46% (n = 436) of boys and 30% (n = 301) of girls are in the midpubertal stage, 4% (n = 36) of boys and 65% (n = 670) of girls are in the advanced pubertal stage, and 0% (n = 0) of boys and 2% (n = 25) of girls are postpubertal.

Seven participants (one individual with anorexia nervosa and six with eating disorder unspecified) were diagnosed with an eating disorder and sought treatment prior to Wave 3 assessment. Seventeen participants were diagnosed and sought treatment prior to Wave 4 assessments including seven participants with anorexia nervosa, nine with eating disorder unspecified, and one participant diagnosed with atypical anorexia nervosa.

Demographics

Table 1 provides the means (sd) for pubertal development and the EDRC, the percent (n) endorsing purging and dieting, and the results of the analysis of variance and chi-square tests examining differences on these measures between girls and boys. Girls scored significantly higher on the PDS and the EDRC and were significantly more likely to report dieting and purging behaviors during both early-to-mid adolescence and young adulthood than boys. Of note, few boys reported engaging in purging behaviors.

Pubertal Development

The results of the analysis of variance and logistic regression analyses for the eating disorder-related variables assessed at Wave 3 are presented in Table 2. For all Wave 3 analyses, none of the interactions were significant so they were dropped from the models. In the final model for the EDRC, higher pubertal development scores in early-to-mid adolescence were significantly associated with higher EDRC scores in late adolescence in both girls and boys (β = 0.024; 95% CI: 0.001, 0.044). Zygosity was not significantly

associated with disordered eating, indicating similar levels of disordered eating across MZ, DZ, and opposite-sex twins.

Higher pubertal development (β = 0.074; 95% CI: 0.023, 0.126) significantly predicted dieting in early-to-mid adolescence. PDS mean scores by dieting were as follows: girls who reported dieting, 14.20 (SD = 2.81), girls who reported never dieting, 13.40 (SD = 3.10), boys who reported dieting, 10.23 (SD = 2.20), and boys who reported never dieting, 10.25 (SD = 2.70). Sex and zygosity were also associated with dieting. MZ twins were less likely to reporting dieting than DZ or opposite-sex twins. However, the number of twins reporting dieting behaviors across the zygosities was somewhat similar with 10% of MZ twins, 16% of DZ twins, and 14% of opposite-sex twins reporting dieting. Pubertal development was not significantly associated with purging behaviors (β = 0.078; 95% CI: −0.001, 0.156) (Table 2). PDS mean scores by purging were as follows: girls who reported purging, 14.23 (SD = 2.64), girls who reported never purging, 13.50 (SD = 3.10), boys who reported purging, 9.70 (SD = 3.00), and boys who reported never purging, 10.25 (SD = 2.70).

The results of the logistic regression analyses for the eating disorder-related variables in young adulthood are presented in Table 3. No interaction terms were significant so they were removed from the models. Pubertal development scores during early-to-mid adolescence did not significantly predict dieting (β = 0.041; 95% CI: −0.029, 0.091) or purging behaviors (β = 0.062; 95% CI: −0.021, 0.145) in young adulthood. Eating disorder diagnosis prior to Wave 4 assessments, dieting at age 16–17, and sex were all significant predictors of dieting at age 19–20. Purging at age 16–17 and sex were significantly associated with purging behaviors in young adulthood. For both dieting and purging, girls were more likely to engage in these behaviors than boys. No differences in zygosity were observed for dieting or purging in young adulthood (Table 3).

Exploring whether there was a differential association between the eating-disorder related variables in late maturers revealed no group differences for dieting (OR = 0.906, 95% CI: 0.541–1.517), purging (OR = 1.319; 95% CI: 0.375–4.631), or disordered eating (η^2 = 0.001, 95% CI: 0.000–0.004) during mid-to-early adolescence or for dieting (OR = 1.208; 95% CI: 0.693–2.106) and purging (OR = 2.530; 95% CI: 0.689–9.297) during young adulthood.

CONCLUSION

We employed a temporally-focused longitudinal design spanning early adolescence to young adulthood to evaluate the association between pubertal development in early-to-mid adolescence and eating disorder-related behaviors in late adolescence and young adulthood in girls and boys. As hypothesized, advanced pubertal development in early-to-mid adolescence predicted EDRC and dieting behaviors in late adolescence. In contrast to our hypothesis, we found no association between pubertal development and purging behaviors. No significant associations between pubertal development and dieting and purging in young adulthood were observed. In addition, those who matured late did now show a differential association with disordered eating compared with those who matured early/on time. Both of these findings are consistent with the hypothesis that the impact of pubertal development on eating disorder-related behaviors is constrained to the developmental period in which pubertal changes are occurring (i.e., adolescence), regardless of the timing of puberty.

Results from previous longitudinal examinations of puberty and disordered eating have been inconsistent.[2, 9, 10, 15, 30] If the impact of puberty on disordered eating is constrained to the adolescent period, as our results suggest, this could account for some of the discrepancies across studies. Current results indicate that when focusing on the impact of general pubertal

development on adolescent outcomes, higher pubertal development scores are in fact significant predictors of disordered eating behaviors. However, this association does not continue into young adulthood. Taken together with previous research, this suggests that early onset and advanced stages of puberty may not be the only periods of vulnerability but the progression through puberty may increase risk for disordered eating.

The exact mechanism by which pubertal development increases vulnerability for disordered eating and dieting has not been elucidated. However, puberty involves many physical changes. For girls, onset involves increasing adiposity and breast development which moves girls further away from Westernized ideals of beauty that emphasize a prepubertal body shape. As adolescence approaches and puberty begins, girls may become more aware of this thin ideal and their deviation from it, leading to increased body dissatisfaction and weight-control attempts which may increase as pubertal development progresses.[16, 31] Additionally, for girls, increased body fatness and obesity during early childhood predicts early onset puberty[32], which could also lead to increased disordered eating and weight-loss attempts. These factors would have an even greater impact for early maturers as many of their peers would still have the idealized prepubertal body shape and size.[33, 34]

For boys, pubertal onset involves the growth of body and facial hair, voice deepening, and increased muscle growth. In contrast to girls, pubertal onset in boys brings them closer to Westernized society's ideal shape for a man (e.g., muscular, hirsute) therefore puberty is thought to be a more positive experience.[35] Our results do not support this assertion. During puberty boys may achieve a more idealized body shape. However, similar to girls, advancing pubertal development and the associated changes may increase feelings of body dissatisfaction and influence other aspects of disordered eating. This would be especially true if pubertal development was occurring earlier than peers and may cause an adolescent's body to “stand out.”

Biological factors might also contribute to the association between pubertal development and disordered eating. Twin studies show that genetic factors account for negligible variance in the liability to disordered eating in prepubertal girls. In contrast, genetic factors account for approximately 60% of the variance in the liability to disordered eating in girls in advanced pubertal stages.[36, 37] The higher levels of reproductive hormones associated with a more advanced pubertal status could moderate this effect. Estradiol is a candidate moderator as the genetic influence on disordered eating in girls with low estradiol levels is minimal and substantial in girls with higher estradiol levels.[38] This has led some researchers to hypothesize that hormone activation at puberty activates the genes responsible for eating disorders in girls.[39, 38] It is unclear how this mechanism would operate in boys.

An important caveat to examining the association between pubertal development and disordered eating is that everyone, typically, will traverse puberty. Thus, pubertal development can only be considered a risk factor for disordered eating when there is variation in pubertal development in the population under study. This is the critical rationale for the necessity of longitudinal studies exploring the impact of puberty on adolescent disordered eating outcomes. Moreover, the current study suggests that it is during this period of development that puberty impacts eating disorder-related behaviors. Therefore, the association between pubertal development and disordered eating is likely complex in nature and a culmination of genetic and environmental factors converging at puberty resulting in increased vulnerability for disordered eating and weight-control attempts. For example, exposure to and internalization of the cultural thin ideal in girls likely plays an important role in reaction to puberty and its physical changes. Girls who internalize this ideal may be more likely to diet or develop disordered eating behaviors as pubertal changes increase adiposity.[34] Perhaps the optimal conceptualization of the role of pubertal development in

risk is that puberty is more related to *when* eating disorder symptomatology develops, not *if* it develops.[2]

The results of this study must be considered within the context of several limitations. First, the eating disorder-related measures were not assessed at Wave 2 so we are unable to directly control for EDRC, dieting, and purging behaviors prior to late adolescence. However, we were able to control for eating disorder diagnosis in those individuals seeking treatment for an eating disorder prior to late adolescence, minimizing the impact of this limitation. Second, participants were not asked to consider dieting and purging behaviors within a specific time frame, therefore it is not known when dieting and purging behaviors began. We additionally had to dichotomize dieting and purging behaviors due to the lower frequency of those behaviors, reducing power. Third, TCHAD represents a single birth cohort in Sweden. Additionally, the prevalence of eating disorders and disordered eating may differ between Sweden and other countries. However, similar rates of eating disorders across the United States, Sweden, and other Scandinavian countries have been suggested.[40, 41] Fourth, a majority of the girls were classified in mid-to-late pubertal stages because they were 13–14 years-old at the time of pubertal development assessment. Thus, our results may reflect an underestimation of the impact of puberty on disordered eating given that those who developed eating disorder symptomatology prior to late adolescence may have experienced a delay in pubertal development. Fifth, the EDI was developed and created for use with female populations. However, it has been shown to function similarly in men and produces similar factor structures, factor loadings and intercorrelations in college men and women.[42, 43] Finally, the prevalence of purging behaviors for both girls and boys was low. This is likely an artifact of the age of the sample, as the age of peak risk for purging in girls is 18.[44]

Our study also has several strengths. First, we used a continuous measure of pubertal development. Although categorizing the PDS based on Tanner's classification scheme has been shown to be reliable and provide “rough” estimates of pubertal development,[45] categorizing this continuous measure may miss subtle differences between the stages and account for discrepancies across studies. Focusing exclusively on pubertal stages, especially during periods when most would be in later pubertal stages, would miss the subtle yet important impact of pubertal development on disordered eating. Second, the population was assessed from early adolescence to young adulthood allowing us to directly examine the impact of pubertal development on eating disorder-related behaviors within the developmental period puberty occurs and to assess if this impact changes across development. Finally, the sample included both girls and boys. Most research examining the role of puberty in disordered eating development has not included a male population. Including girls and boys allowed us to compare the sexes and determine if pubertal development presents the same association with and risk for disordered eating in boys that it does for girls.

Current findings suggest that higher pubertal development scores are predictive of disordered eating during adolescence in girls and boys. However, this association is constrained to the adolescent period as pubertal development during adolescence is not associated with disordered eating in young adulthood. Although the timing of pubertal onset or pubertal status compared to peers (e.g., early, late, on time) may increase risk for disordered eating, it appears that the progression through puberty also increases risk. It is important for clinicians to be aware that pubertal development increases risk for disordered eating during adolescence. Although this might be common knowledge for girls, it is important to have awareness of the risk for boys. Future research should further address the complex association between pubertal development and disordered eating by examining

possible mediating factors such as internalization of the cultural thin ideal and reproductive hormone levels.

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Table 1

Results from analysis of variance and chi-square tests comparing girls and boys on pubertal development and eating disorder-related variables

	Girls Mean (sd)	Boys Mean (sd)	Test-statistic *F* (*p*-value)
Pubertal Development at Age 13–14	13.6 (3.0)	10.2 (2.7)	666.00 ($p < .001$)
Range	5.0 – 20.0	5.0 – 18.0	
EDRC at Age 16–17	9.1 (10.0)	3.6 (5.0)	250.55 ($p < .001$)
Range	0 – 58	0 – 37	

	% (*n*)	% (*n*)	χ^2 (*p*-value)
Dieting at Age 16–17	21.9 (236)	4.2 (41)	137.22 ($p < .001$)
Purging at Age 16–17	8.0 (87)	0.3 (3)	73.23 ($p < .001$)
Dieting at Age 19–20	36.0 (309)	7.4 (43)	152.51 ($p < .001$)
Purging at Age 19–20	12.4 (107)	0.7 (4)	67.10 ($p < .001$)

Note. sd = standard deviation. EDRC = Eating Disorder Risk Composite. % = percentage of participants endorsing dieting or purging.

n = number of participants endorsing dieting or purging.

Table 2

Chi-square results from analysis of variance and logistic regression models with eating-related variables at age 16–17 as the dependent variable and pubertal development, sex, and zygosity as the independent variables. Generalized estimating equation modeling corrections applied to account for non-independence of the data.

Independent Variables	EDRC χ^2	Dieting χ^2	Purging Behaviors χ^2
Pubertal Development	6.04 *	8.45 **	4.16
Eating Disorder Diagnosis	0.17	0.49	1.56
Sex	82.73 **	59.39 **	39.21 **
Zygosity	4.06	10.41 *	3.55

Note. EDRC = Eating Disorder Risk Composite.

* $p < 0.05$

** $p < 0.01$

Table 3

Chi-square results from logistic regression models with eating-related variables at age 19–20 as the dependent variable and pubertal development, sex, and zygosity as the independent variables. Generalized estimating equation modeling corrections applied to account for non-independence of the data.

Independent Variables	Dieting χ^2	Purging Behaviors χ^2
Pubertal Development	2.55	2.24
Eating Disorder Diagnosis	5.36*	4.01
Dieting at Age 16–17	100.41**	--
Purging at Age 16–17	--	26.24**
Sex	45.01**	27.54**
Zygosity	1.47	0.27

* $p < 0.05$

** $p < 0.01$